

عنوان مقاله:

Genetic markers and smoking status

محل انتشار: سومین کنگره ملی دخانیات و سلامت (سال: 1397)

تعداد صفحات اصل مقاله: 2

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خلاصه مقاله:

Smoking is one of the most harmful behaviors linked with harmful health conditions in smokers and passive smokers. Smoking has "stages: initiating, current smoking and smoking cessation. This behavior is a multistep process with genetic influences. Current review focuses on loci associated with smoking behavior and predictive epigenetic biomarkers for smoking status. NTRKr is associated with smoking initiation and dependence. BDNF binds with NTRK and reinforces nicotine dependence and smoking behavior. The tobacco and genetics (TAG) consortium reports variants at the BDNF locus influence smoking initiation. A region on chromosome 16 (16qY6) including the nicotinic receptor subunit genes CHRNA6, CHRNAM and CHRNBF (encoding NAChr receptors) was associated with cigarettes consumed per day (CPD), a measure of smoking dependence. The TAG and european network for genetic and genomic epidemiology (ENGAGE) consortia also identified the CHRNAF-CHRNBr cluster on chromosome ApII, as well as loci found near CYPYAF on 19g1^w and in a region containing noncoding RNAs on chromosome 1.9Y^w and 1.9Y^A. as associated with CPD with unknown function. Association of CHRNAF with smoking behavior was reported previously in a candidate gene study and is consistent with the gene's high expression in dopamine-releasing neurons (which have a key role in smoking). For smoking cessation, the TAG consortium identified a region on chromosome 9 near the gene encoding dopamine β-hydroxylase (DBH) which catalyzes the conversion of dopamine to norepinephrine. Polymorphisms of cytochrome pF۵۰۲A۶ (encoded by CYPYA۶) strongly influence the catabolism of nicotine into inactive metabolites, and individuals with rapid metabolism require higher levels of smoking to maintain the same nicotine level than do individuals whose genotypes confer slower metabolism. Epigenome-wide association studies (EpWAS) in Europe revealed that LRRNr (among CpG cites) had increased expression and methylation and could be a biomarker for smoking status. EpWAS results in comparing nonsmokers and smokers show " different loci on the AHRR gene (chromosome Δ) (P-values from 10-109 to 10-1F), F CpG sites in a non-annotated region on chromosome Y (YqWY.I), a sites on chromosome I (including F sites on GFII and I on GNGIY), I CpG site on chromosome 19 (FYRLT), 1 CpG site in a non-annotated region on chromosome 5 (SpY1.TT) and T CpG sites on

chromosome II (KCNQIOTI), IF (RARG) and IF (ADCY9), respectively. Hypomethylation at CpG sites on AHRR and ... GFII in new-born cord blood related is related to maternal smoking. Several hyper

کلمات کلیدی: Smoking, Gene, Expression, Biomarker, Epigenetics

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